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DEVELOPMENT OF GLUTEN-FREE CEREAL PRODUCTS

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Celiac disease

Coeliac disease or in the U.S. celiac disease (also known as non-tropical sprue, gluten-sensitive enteropathy, celiac sprue, idiopathic steatorrhea, primary malabsorption, Gee-Herter disease, gluten-induced enteropathy, adult celiac disease) is a condition where the person's body reacts to the protein fraction gluten (Cooke & Asquith 1974). The commonly encountered reaction to gluten by sufferers of the coeliac disease are outlined in Table. 1.

Table 1: Symptoms (and related signs) of coeliac disease (Feighery, 1999).

Infancy (0-2 years):

Diarrhoea (miserable, pale)
Abdominal distension (enlarged abdomen)
Failure to thrive (low weight, lack of fat, hair thinning)
Anorexia, vomiting
Psychomotor impairment (muscle wasting)

Childhood:

Diarrhoea or constipation
Anaemia
Loss of appetite (short stature, osteoporosis)

Adulthood:

Diarrhoea or constipation
Anaemia
Aphthous ulcers, sore tongue and mouth (mouth ulcers, glossitis, stomatitis)
Dyspepsia, abdominal pain, bloating (weight loss)
Fatigue, infertility, neuropsychiatric symptoms (anxiety, depression)
Bone pain (osteoporosis)
Weakness (myopathy, neuropathy)
Epidemiology

Coeliac disease is more common in Ireland than anywhere else in the world. It is particularly prevalent in the West of Ireland. Conservative estimates in Ireland consider that approximately ten thousand people suffer from undiagnosed coeliac disease, who regularly feel unwell without ever knowing why. Factors suggested as influencing the high incidence of coeliac disease in the west of Ireland include the environment, intermarriage, dependence on the potato together with infant feeding habits. However, it should be noted that none of these factors have been proven, as yet (Stevens, 1980). Research has also shown that coeliac disease has the highest incidence among the Caucasians of Western and Southern Europe. Coeliac disease is also common in Australia, North America and Latin America as a direct result of emigrated descendents from Europe (Stevens, 1980). While coeliac disease is more prevalent in the previously mentioned societies, it should be noted that the condition also occurs in other cultures (Barry *et al.*; 1974).

The iceberg model

The iceberg is a common model used to explain the epidemiology of coeliac disease (Visakorpi, 1997). An iceberg model is shown in Figure 1. According to Stern (1992) the tip of the iceberg is formed by patients with overt disease who have just been diagnosed by biopsy demonstrating a flat mucosa. The lower part of the tip is formed by patients who have been recently diagnosed and who are now living-gluten free and show a normal mucosa. Below the waterline there is a big group of "silent" cases, which have not been identified and have a flat small intestine mucosa. They may remain undiagnosed because the condition has no symptoms (Feighery 1999). Just at the bottom there is a small group of patients with latent coeliac disease showing a normal mucosa while taking gluten.

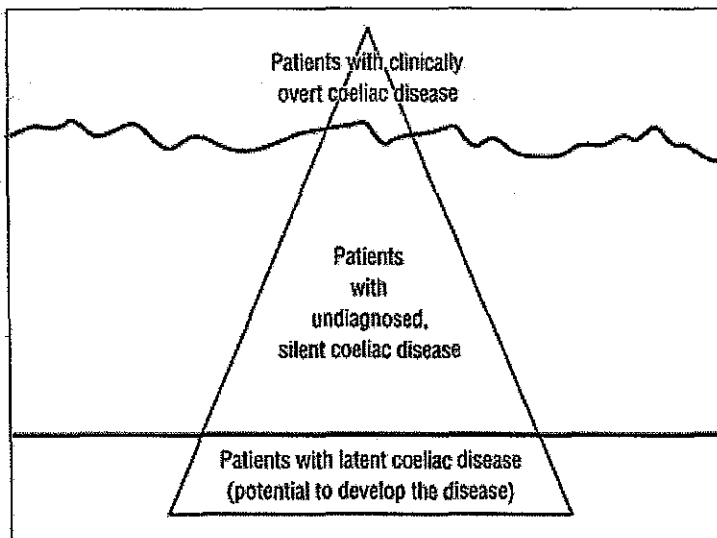


Fig. 1: Iceberg model depicting prevalence of coeliac disease (Feighery, 1999).

Disease Mechanisms

The entire surface of the small intestine is covered in a layer of epithelial cells, that are cyclically renewed every four days. As the cells mature, they increase their enzyme activity and transport capabilities. Many of the enzymes that complete the digestion of food molecules are associated with the microvilli, including proteases and disaccharidases (e.g. lactase). On eating wheat, the coeliac undergoes an immunological response, localized in the small intestine, which destroys mature absorptive epithelial cells on the surface of the small intestine. Destruction of the cells decreases the heights of the villi (Figure 2). Increased cell division, due to cell destructions, results in the proliferation of immature epithelial cells. The mucosa assumes a flat appearance with the disappearance of the villi and the thickening of the surface, which results in decreased absorptive surface area, diminished enzyme activities and transport activities which contribute to malabsorption and affects all systems of the body. The immunologic response to gluten may also occur secondarily in other bodily tissues, an example being dermatitis herpetiformis (Pruessner, 1998).

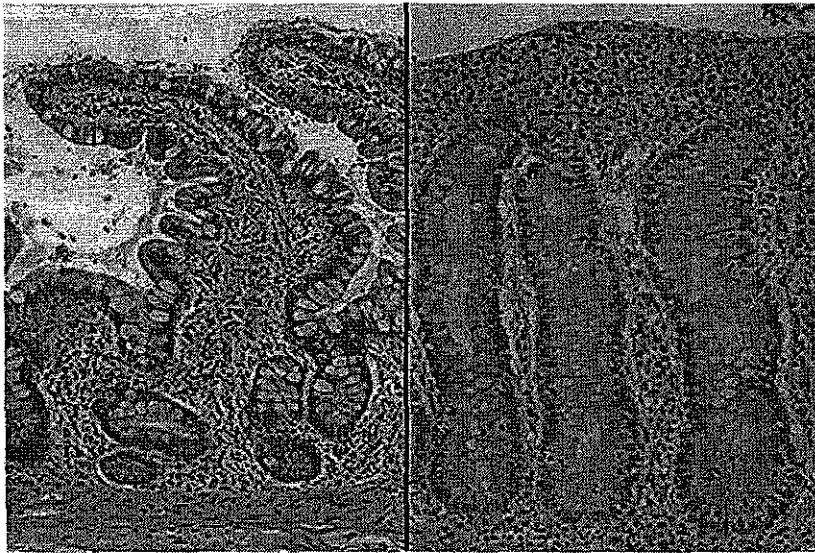


Fig. 2: Normal small intestinal mucosa is seen at the left, and mucosa involved by celiac sprue at the right.

In a study by De Ritis *et al.*; (1988) it was shown that specifically the gliadin portion of wheat protein, and prolamines (alcohol soluble proteins) in rye, barley and oats are the toxic fraction.

Possible Causes

According to Murray (1999) coeliac disease is the end result of three processes that culminate in intestinal mucosal damage: genetic predisposition, environmental factors and immunologically based inflammation.

Diagnosis

The most important test to examine the presence of coeliac disease is to carry out a biopsy, from the surface of the small intestine. If the biopsy is positive the internal surface is checked again after the patient has been adhering to gluten free diet (Stern, 1992). A number of Blood Screening Tests can also be used for an indication of coeliac disease. Such as: Alkaline phosphatase level, Anaemia, Cholesterol and low-density cholesterol levels, Aspartate amino-Transferase level and Plasma protein-Albumin level.

Cereal Chemistry

Wheat, rye and barley are members of the grass family and are quite closely related to one another according to various schemes of plant classification (taxonomy). The deleterious proteins are gliadins (wheat), hordeins (barley), secalins (rye), and possibly avidins (oats) (Murray, 1999, Kasarda, 2001). The proteins are complex storage proteins that share some common repetitive sequences. The exact peptide sequences involved have not been identified precisely, although peptides rich in glutamines and prolines are potent activators of the immune response in coeliac disease (Murray, 1999). Kasarda (2001) recommended that corn and rice was safer for celiacs, then members of the grass family. Such grasses include sorghum, millet, teff, ragi, and Job's tears.

Treatment

According to Taylor (1999) the avoidance of wheat, rye, barley and oats and all products made from these grains is the only treatment for coeliac disease. Treatment with gluten-free diet results in significant improvement of the intestinal mucosa and its absorptive function.

Foods not allowed in a gluten-free diet:

Any bread, cereal or other food made with wheat, rye, barley, triticale, dinkel, kamut, spelta and oat flour or ingredients and by-products made from those grains.

Processed foods that contain wheat and gluten-derivatives such as thickeners and fillers like hot dogs, ice cream, salad dressings, canned soups, dried soup mixes, non-dairy creamers, processed cheese, cream sauces, and hundreds of other common foods.

Medications that use gluten to bind a pill or tablet together.

Bread made from nominally gluten-free flours, which are based on wheat starch contain small amounts of gliadin. Regular intake of these products may explain why some patients fail to respond to gluten-free diet (Ellis *et al.*; 1998).

Related conditions

As coeliac disease is an autoimmune disease, it follows that there are other autoimmune diseases associated with it. This is because coeliacs are part of a group genetically disposed to autoimmune diseases (Brody, 1997). Coeliac patients have increased gastrointestinal permeability and demonstrate whole body effects of food allergy, including brain dysfunction, arthritis and inflammatory lung disease. Diabetes, thyroid disease, purpura, anaemia, rheumatoid arthritis, sacroileitis, sarcoidosis, vasculitis, lung disease, myositis, eye inflammation, and schizophrenia are all linked to gluten intolerance.

Gluten-free – Codex Alimentarius

The Codex Alimentarius Commission of the World Health Organization (WHO) and the Food and Agricultural Organisation (FAO) stated in 1981 and in 2000 with a draft revise standard for gluten-free foods that so-called “gluten-free” foods are described as: a) consisting of, or made only from ingredients which do not contain any prolamins from wheat or all *Triticum* species such as spelt (*Triticum spelta* L.), kamut (*Triticum polonicum* L.) or durum wheat, rye, barley, oats or their crossbred varieties with a gluten level not exceeding [20 ppm]; or b) consisting of ingredients from wheat, rye, barley, oats, spelt or their crossbred varieties, which have been rendered “gluten-free”; with a gluten level not exceeding [200 ppm]; or c) any mixture of two ingredients as in a) and b) mentioned with a level not exceeding [200 ppm].

In this context the WHO/FAO standard “gluten” was defined as the protein found in wheat, triticale, rye, barley, oats, or in crossbred varieties of them and derivatives thereof, to which some people are intolerant and that is insoluble in water and 0.5M NaCl. Prolamins are defined as the fraction from gluten that can be extracted by 40-70% of ethanol. The prolamins of wheat is gliadin, from rye is secalin, from barley is hordein and from oats is avenin. It is however an established custom to speak of gluten sensitivity. The prolamins content of gluten is generally taken as 50%.

Irish Market of gluten free cereal products

A wide range of gluten free cereal products ranging from breads to pizza and biscuits were collected and subjected to a range of physical and sensory tests. The former embraced texture profile analysis, moisture determination, crumb density, crust and crumb colour. Particular attention focused on the rate of staling. Sensory testing was also carried out by assessing the appearance (whole loaves, and slice appearance), crumbliness (in relation to spreadability of butter), mouthfeel, overall texture and flavour. Overall it was found that the gluten free products were of inferior quality and

very often showed off-flavours. The structure of the products was mostly crumbly and very dry. The majority of bread products are sold as part baked products, which need to be reheated and are not suitable for sandwiches.

Gluten free biscuits

Biscuits are very popular products all over the world, the vast combinations of texture and taste have given biscuits and cookies a universal appeal. The three main ingredients are wheat flour, fat and sugar. In different combinations, they form the basis of a full range of biscuit products. In gluten free biscuits the wheat flour, which originates from soft winter wheat needs to be replaced by other ingredients. These ingredients need to replace not only the starch, which is delivered by the wheat flour but also the protein fractions. The advantage in developing gluten free biscuits is that gluten network formation is unwanted in a lot of biscuit products. The objective of this study was to develop a biscuit, which is as close as possible to wheat based products. In our study we chose to work with short dough biscuits. The effect of a variety of starches ranging from corn, soya, millet, rice and potatoes were combined with different types of fat (palm oil, cream powder, microencapsulated high fat powder and low fat dairy powder). The dough characteristics as well as the texture, colour and moisture, dimensions and sensory attributes were evaluated. It was found that combinations of rice, potato, corn and soya with high fat powders produced biscuit doughs, which are sheetable, and biscuits of comparable quality to the control (wheat biscuits).

Gluten free pizza

Pizza has become one of the most popular convenience products. Currently the quality of gluten free products is poor and is closer to a cake product than a wheat dough pizza. The quality criteria for a good quality pizza would be, that the dough has to be sheetable, rises on proving and holds the gas produced by the yeast, as well as having good textural and sensory attributes. By combining a variety of starches, protein sources with a microencapsulated high fat powder it was possible to fulfil all the requirements stated above. Tests such as dough stickiness and hardness, volume, colour and pizza texture confirmed that it is possible to produce a gluten free pizza product with similar attributes to the wheat based control. The influence of the various ingredients on the dough rheology of the optimised recipe were tested using an oscillation test. This test was performed at ten frequencies ranging from 0.1 to 10 Hz. The parameters measured were phase angle, elastic modulus and viscous modulus. From these measurements it was very clearly seen that in the cornstarch system the biggest increase in elasticity was achieved, when guar gum was combined with high fat powder.

Gluten free breads

The replacement of gluten in bread dough is one of the biggest challenges, when working with gluten free cereal products. This is reflected when evaluating the

products, which are currently on the market, which are mostly of inferior quality. The wheat protein namely gluten has such a wide variety of tasks in bread making that a wide range of ingredients are needed to achieve a good quality product without it. The majority of the gluten free flours as well as gluten free products currently on the market are wheat starch based and can therefore threaten the health of a coeliac patient, due to the very small amount of gluten that might be still present. The approach we have taken is to only work with starches, which are listed as gluten free, such as, corn, potato, soya, buckwheat and rice. These starches were then combined with gums and dairy ingredients to produce a gluten free bread of similar quality to wheat bread. The loaf volume, moisture, water activity (a_w) and colour of the baked goods were evaluated. The texture profiles and the extent of staling during storage of these baked goods were also determined. Sensory evaluation was also carried out with members of the coeliac association of Ireland as well as normal taste panels. The image characteristics were also determined by using an image analysis system. Overall the products, which have been developed so far are superior to any of the products on the market and are comparable to their wheat base counterparts.

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